Objectives Reversion of this nucleotide to the ancestral type, –220A, co-occurs with severe deficit in higher brain cognitive functions.
Aims In the current study, we compare the pattern of protein binding between –220C and –220A.
Methods Antibodies reactive against transcription factors CREB, USF, and c-Myc were used to identify the specific proteins involved in complexes with DNA using electrophoretic mobility shift assay (EMSA).
Results Significant increase was observed in the overall protein complexes binding to the –220 C allele vs. –220A. The transcription factors, CREB, USF, and c-Myc, were differentially bound to –220C, represented by supershifts.
Conclusions We propose that differential binding of CREB, USF, and c-Myc to CALR nucleotide –220C may be linked with the evolution of higher brain functions in human.
Disclosure of interest The author has not supplied his/her declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.1694

EV712

Neurofarmagen® testing and drug side effects: An evaluation of its use among a real-world case series

F. Oliva [1,*], A. Portigliatti Pomeri [2], G. Nibbio [3], M. Giuseppe [2]
[1] *University of Turin, Department of Biological and Clinical Sciences, Orbassano, TO, Italy*
[2] *University of Turin, Department of Neurosciences "Rita Levi Montalcini", Orbassano, TO, Italy*
[3] *University of Turin, Department Biological and Clinical Sciences, Orbassano, TO, Italy*
* *Corresponding author.*

Introduction Various pharmacokinetic and pharmacodynamics features have proven to be involved in the development of drug-induced side effects in psychiatry and thus pharmacogenetic profiling should be considered during drug selection to avoid the onset of side effects.
Aim To explore the usefulness of Neurofarmagen® testing in clinical practice by evaluating whether the genetic profile given by the tool could properly explain the onset of side effects during antipsychotic treatment.
Methods The pharmacogenetic profile of ten patients having a history of side effect appeared during to specific a psychopharmacologic treatment was determined by Neurofarmagen® testing tool. The relationship between genetic profile and side effects was evaluated and classified.
Results Sixty percent of the sample showed a genomic alteration related to a increased likelihood of having any side effects, one half of which presented pharmacokinetic alteration (slow or intermediate phenotype for the implicated cytochrome) whereas the other half had a pharmacodynamic gene variant (related to dopamine or serotonin pathway).
Conclusion the Neurofarmagen® testing tool may be useful in the clinical practice in order to avoid drug-induced side effects.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.1697

EV713

Psychiatric manifestations of Niemann-Pick type C disease – two case reports

J. Rebelo*, M. Oliveira, P. Nunes
Centro Hospitalar de São João, E.P.E., Psychiatry, Porto, Portugal
* *Corresponding author.*

Introduction Niemann-Pick type C disease (NPCD) is a rare metabolic illness, with autosomal recessive inheritance. NPCD has a heterogeneous presentation, with non-specific psychiatric symptoms, mostly affective and psychotic features and also cognitive deficits.
Objectives and methods We present the case reports of two brothers with an adolescent-adult onset and discuss the evolution of their neuropsychiatric manifestations.
Results The patients have now 35 and 31 years old and the youngest was the first to develop clinical manifestations of the disease. From 16 years old, he developed unspecified neurological impairment with gait imbalance. In the next years, the neurologic manifestations exacerbated, with dysarthria, ataxic gait, and his academic performance declined. With 24 years old, he presented acute psychosis, with unstructured delusion and auditory hallucinations. The acute psychotic symptomatology remitted with olanzapine but he revealed social withdrawal, apathy and progressive cognitive decline that persist until now. His brother, whose diagnosis was made in the course of the family genetic study, developed the first signs of the NPCD with 19 years old. He presented neuropsychiatric compromise, with impaired learning, social isolation and insomnia. They are receiving specific treatment with miglustat and symptomatic treatment for the psychiatric manifestations.
Conclusions NPCD is a rare metabolic disease, with neuropsychiatric compromise. No general psychopathological profile has been associated to NPCD. Sometimes psychiatric symptoms dominate the initial clinical presentation, with neuro-visceral signs appearing later. An atypical psychiatric symptomatology should be extensively investigated in order to exclude organic causes, including metabolic diseases like NPCD.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.1698

EV714

Psychiatric disturbances in a patient with melas syndrome: A case report

M.C. Rizza [1,*], S. Di Marco [1], C. Delicato [1], C. Vecchi [1], C. Gramaglia [1], P. Prosperini [2], R. Cantello [1], P. Zeppegno [1]
[1] *Università del Piemonte Orientale, Traslational Medicine, Novara, Italy*
[2] *Azienda Ospedaliera Universitaria "Maggiore della Carità", Traslational Medicine, Novara, Italy*
* *Corresponding author.*

Introduction Mitochondrial disorders of energetic metabolism (MD) represent a heterogeneous group of diseases manifesting at any age and its one of a number of mitochondria syndromes that share the common characteristics of encephalopathy and myopathy. The clinical expression of MELAS (Mitochondrial Myopathy, Encephalopathy, Lactic Acidosis and Stroke-like episodes) is highly variable and ppsychiatric symptoms are rarely reported in literature even if are more common in MELAS syndrome than in the general population.
Objective The first aim of the study is describing the clinically observed primary psychiatric symptoms in a patient affected by MELAS syndrome admitted to the Psychiatric ward. The second aim is to go back over the diagnostic process, which led, from the uncommon psychiatric symptoms and signs to the final genetic diagnosis of MD.
Methods and results We report the case of a 44-year-old male with MELAS in whom psychiatric symptoms preceded the establishment of the clinical diagnosis for several months. Diagnosis was initially based on the neuroimaging and metabolic findings and subsequently confirmed with genetic analysis.
Conclusions In case of aggressive and paranoid behaviour with delusions of persecution and disorganised behaviour mmitochon-

https://doi.org/10.1016/j.eurpsy.2016.01.1699 Published online by Cambridge University Press

drial disorders deserve consideration as part of the differential diagnosis, especially if there is suspected involvement of other organ groups or positive family history of MD. There is no specific consensus approach for treating MELAS syndrome. Management is largely symptomatic and should involve a multidisciplinary team.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.1699

EV716

Serine racemase in inhibitory neurons at striatum and it might be involved in schizophrenia's pathophysiology with D1 and D2 receptors

S. Takagi[1,*], D. Balu[2], J. Coyle[2]
[1] *Tokyo Medical and Dental University, Psychiatry, Tokyo, Japan*
[2] *Harvard Medical School, Department of Psychiatry, Boston, USA*
[*] *Corresponding author.*

Introduction There is substantial evidence that hypofunction of the *N*-methyl-D-aspartate receptor (NMDAR) is a core pathophysiological mechanism underlying schizophrenia. D-serine and serine racemase (SR) (NMDAR co-agonist and it's producer) are thought to be involved in schizophrenia's pathophysiology as NMDAR function moderators. Our laboratory showed that excitatory neuron specific SR knock out (SRKO) mice still have just 50% reduction of SR whereas full SRKO mice had no SR. Furthermore D-serine and SR are found in inhibitory neurons not only in excitatory neurons with immunohistochemistry methods. Because NMDAR has excitatory functions, the existence of D-serine and SR in inhibitory neurons and their functions are of interest.
Aims To elucidate the existence and roles of D-serine and SR in inhibitory neurons.
Methods Inhibitory neuron marker, GAD65, specific conditional SRKO (GAD65 SRKO) mice were made by Cre-lox recombination method. The GAD65 SRKO mice were analyzed by HPLC for D-serine concentration, western blotting for SR expression, immunohistochemistry for SR positive cell's character identification and behavioral testing.
Results GAD65 SRKO had about 50% reduction of SR in striatum but no reduction in hippocampus and frontal cortex. D-serine of GAD65 SRKO mice was not different from WT mice. Immunohistochemistry works revealed SR is in medium spiny neuron of striatum and has colocalization with DARRP-32, D1 receptor, and D2 receptor.
Conclusions SR is expressed in inhibitory neurons at least in striatum. It might be involved in schizophrenia's pathophysiology because it colocalizes with D1 and D2 receptors.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.1701

Geriatric psychiatry

EV717

Catatonia and dementia

M. Almeida[*], C. Gama Pereira
Centro Hospitalar Baixo Vouga, Departamento de Psiquiatria e Saúde Mental, Aveiro, Portugal
[*] *Corresponding author.*

Introduction Catatonia, described by Kahlbaum in 1874, is usually seen as a type of schizophrenia, but it can also occur in a wide range of other psychiatric/organic disturbances. There is a documented association between dementia and catatonia, in all phases of cognitive impairment.
Aims Literature review and discussion about Catatonia, regarding a case report.
Methods Clinical interviews and literature review in PUBMED database.
Results (case report) Female patient, 89 years old, without psychiatric history, was diagnosed with dementia 5 months prior to episode. On admission, she presents with prostration, mutism and refusal to eat/drink. Laboratory studies were normal and TC-CE shows signs of an old stroke in left temporo-parietal region and diffuse signs of ischemic leucoencephalopathy. At psychiatric evaluation, she was stuporous, unreactive to pain, mute, not following verbal commands, keeping her eyes closed and resisting attempts to open her eyelids. She had global rigidity, axial and limbs, and maintains the postures the examiner puts her into for long periods. She was already given chlorpromazine, without improvement. Then she takes diazepam 10 mg iv, with remission of the state.
Conclusions Although catatonia usually presents with drama, clinicians often forget to consider it in differential diagnosis, probably because of its traditional association with schizophrenia. A promptly diagnostic is crucial to provide adequate treatment, avoiding drugs that can worsen/perpetuate the clinical state. Some authors even support the idea that motor features associated with end-line dementias may correspond to lorazepam-responsive catatonia, in which treatment may have a tremendous impact worldwide.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.1702

EV718

Mini-Mental State (MMS) evaluation of dementia in psychiatric patients admitted to a long stay ward

N. Amel[*], L.P. Isabelle, G. Aurelie, H. Marc, A. Mohammed, I. Ziad
USLD La Roseraie, EPS Maison-blanche, Seine-Saint-Denis, Neuilly-Sur-Marne, France
[*] *Corresponding author.*

Normal 0 21 false false false FR X-NONE X-NONE MicrosoftInternetExplorer4.

MMS scores for 41 psychiatric patients were analyzed at admission and regularly throughout their stay.
Results Their average age at admission was 65.7. Thirty-six patients had a diagnosis of chronic psychosis, two with bipolar disorders, one with frontotemporal dementia, two with Korsakoff syndrome.
At admission, 21 (51%) patientsshowed mild cognitive deterioration (score = 18–26), 12 (29%) moderate deterioration (12–17), 6 severe deterioration (0–11), 2 had normal scores (27–30). Over the following years, 28 patients were reassessed:
– 12 (42%) were stable, 7 (25%) had a fluctuating score, 5 (18%) improved;
– 4 (14%) deteriorated over their successive MMS evaluations;
– age, socio-cultural level and psychiatric diagnosis were not associated with change in MMS scores;
– average change between initial and final assessment was +6.0 points for patients with improved score, –7.75 for those showing deterioration;
– 1.28 for those with fluctuating scores, –1.0 for stable patients.
Analysis Unstable psychiatric disorders associated with somatic pathologies influenced MMS scores for all patients, particularly for those with MMS deterioration or fluctuation even if this phenomenon could also be observed to a lesser extent in stable patients. By contrast, patients whose MMS scores improved over